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Maternal effects and population regulation: maternal density-induced reproduction suppression impairs offspring capacity in response to immediate environment in root voles *Microtus oeconomus*

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Summary

1. The hypothesis that maternal effects act as an adaptive bridge in translating maternal environments into offspring phenotypes, and thereby affecting population dynamics has not been studied in the well-controlled fields.

2. In this study, the effects of maternal population density on offspring stress axis, reproduction and population dynamics were studied in root voles (*Microtus oeconomus*). Parental enclosures for breeding offspring were established by introducing six adults per sex into each of 4 (low density) and 30 adults per sex into each of another 4 (high density) enclosures. Live-trapping started 2 weeks after. Offspring captured at age of 20–30 days were removed to the laboratory, housed under laboratory conditions until puberty, and subsequently used to establish offspring populations in these same enclosures, after parental populations had been removed. [Correction added on 8 January 2015 after first online publication: '10–20 days' has been changed to '20–30 days.'] Offspring from each of the two parental sources were assigned into four enclosures with two for each of the two density treatments used in establishing parental populations (referred to as LL and LH for maternally unstressed offspring, assigned in low and highdensity, and HL and HH for maternally stressed offspring, assigned in low and high density). Faecal corticosterone metabolites (FCM) levels, offspring reproduction traits and population dynamics were tested following repeated live-trapping over two seasons.

3. Differential fluctuations in population size were observed between maternally density-stressed and density-unstressed offspring. Populations in LL and LH groups changed significantly in responding to initial density and reached the similar levels at beginning of the second trapping season. Populations in HL and HH groups, however, were remained relatively steady, and in HL group, the low population size was sustained until end of experiment. Maternal density stress was associated with FCM elevations, reproduction suppression and body mass decrease at sexual maturity in offspring. The FCM elevations and reproduction suppression were independent of offspring population density and correlated with decreased offspring quality.

4. These findings indicate that intrinsic state alterations induced by maternal stress impair offspring capacity in response to immediate environment, and these alterations are likely mediated by maternal stress system. The maladaptive reproduction suppression seen in HL group suggests intrinsic population density as one of ecological factors generating delayed density-dependent effects.

Key-words: ecological stressor, environmental mismatch, glucocorticoid metabolites, maternal matching, none-genetic phenotypic effects, population self-regulation

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Introduction

Small mammal populations in the Northern Hemisphere often fluctuate cyclically with population peaks occurring every 3–5 years, and such fluctuations have inspired considerable studies in ecology (see review by Krebs & Myers 1974; Krebs 1996, 2013; Batzli 1996), attempting to identify the potential causes and mechanisms undying these phenomena, since the publication of Elton's influential paper (Elton 1924). Accumulated hypotheses and empirical findings suggest involvement of multiple ecological factors acting at various phases of the population cycle, including those extrinsic or intrinsic to the populations. The major extrinsic factors include predation (Korpimäki & Krebs 1996; Lambin, Petty & Mackinnon 2000; Hanski *et al*. 2001; Oli 2003) and resource quality (Batzli 1986; Turchin & Batzli 2001). The intrinsic factors that were originally considered include genotypic and phenotypic changes in populations associated with density changes (Krebs 1978; Boonstra & Boag 1987; Boonstra 1994; Boonstra & Hochachka 1997; Tkadlec & Zejda 1998). While the roles of extrinsic factors have received wide support from field studies, considerable controversies exist concerning whether intrinsic factors are able to generate any significant effects, particularly in the sustained low phase of the population cycle. Indeed, the roles of genetic behavioural polymorphisms have never been demonstrated empirically and density-dependent variations in population age structure appear to have limited effects on population dynamics (Boonstra & Boag 1987; Boonstra & Hochachka 1997; Tkadlec & Zejda 1998).

Life-history traits relating to growth and reproduction are critical determinants in population dynamics (Oksanen *et al*. 2007). In vole populations, body mass and age at maturation vary considerably along with the phase fluctuations of population cycles (Oli & Dobson 1999). Individuals in increasing populations are typically larger, start reproduction earlier in spring than those in declining populations (Krebs & Myers 1974; Boonstra & Krebs 1979; Boonstra 1994). These life-history patterns of phenotype variations can also be seen in other microtinae fluctuating populations (Boonstra & Boag 1992; Mihok & Boonstra 1992; Prevot-Julliard *et al*. 1999; Ergon *et al*. 2001) and indicate that maternal modifications of offspring reproduction traits may represent one intrinsic process under which maternal environment can influence population dynamics. Thus, identification of the physiological systems that mediate the translation of maternal environmental signals into offspring phenotypes would be critical for understanding the role of intrinsic factors in the regulation of population dynamics (Sheriff & Love 2013).

Most ecological factors known to influence population dynamics are normally also modulators of individual stress responses in the population. Predator presentation (Boonstra & Boag 1992; Boonstra *et al*. 1998; Sheriff, Krebs & Boonstra 2009; Clinchy, Sheriff & Zanette 2013), resource depletion (Jeanniard du Dot, Rosen & Trites 2009), as well as increase in population density (Rogovin *et al*. 2003; Bian *et al*. 2011; Creel *et al*. 2013) each has been reported to elevate levels of glucocorticoids (GC), a critical hormone in stress response. Stress, particularly chronic stress, plays a critical role in determining the phenotypes of numerous life-history traits at physical, physiological and behavioural levels (see review by Johnson *et al*. 1992 and Crespi *et al*. 2013). Lines of laboratory evidence from rat studies indicate that maternal stress is associated with a series of plasticity in offspring stress axis, the effects mediated by elevations in maternal GC levels (Welberg & Seckl 2001; Owen, Andrews & Matthews 2005). Based on these findings, maternal stress axis has been proposed as one of plausible physiological systems for translation of maternal environment into offspring phenotypes (Boonstra & Boag 1992; Boonstra *et al*. 1998; Love, McGowan & Sheriff 2013; Sheriff & Love 2013). However, several field studies failed to detect maternal effects, when only the proximate effects on offspring phenotype were examined (Ergon *et al*. 2001; Ergon, Lambin & Stenseth 2001; Klemola, Korpimaki & Koivula 2002; Banks & Powell 2004; but see Boonstra & Boag 1987) within a relatively short period. Therefore, it would be necessary to explore maternal effects under a relatively long life-history background in the well-controlled field.

In the present study, we have investigated the effects of maternal stress on offspring GC activity, reproduction and population dynamics via manipulation of population density in root voles (*Microtus oeconomus*). Parental enclosures with high (30 voles per sex per enclosure) and low (6 per sex per enclosure) population densities were initially established to breed offspring. Subsequently, offspring enclosures with the same 2 density treatments were established for offspring from each parental source. Offspring population density trajectories, GC activity, reproductive traits and overwinter survival in each of four treatment groups were measured following live-trapping across two breeding seasons. The aim of this study was to test whether the plasticity of offspring stress axis, originally seen in laboratory animals and recently in snowshoe hares (Sheriff, Krebs & Boonstra 2010b) can also be manifested in this wild-living species, and if it does, whether such plasticity is associated with alterations in offspring reproduction normally seen in stressed animals, and finally whether such alterations impact offspring population dynamics. We hypothesize that, if the plasticity occurs and persists, density-induced maternal stress would alter offspring GC activity and reproduction independent of the environment in which the offspring live. The adaptive potential of such alterations would depend on the degree at which maternal effects predict offspring living environment (Love, McGowan & Sheriff 2013; Sheriff & Love 2013).

Materials and methods

ROOT VOLES IN THE STUDY AREA

Our study was conducted at Haibei Alpine Meadow Ecosystem Research Station, Menyuan County, *c*. 155 Km N of Xining, the capital city of Qinghai province, People's Republic of China (37°37′N, 101°12′E). The area is a secondary vegetation type meadow with dense and high plant leaf-layers. The major plant species include *Poa* sp., *Elymus nutans*, *Kobresia humilis*, *Potentila fruticosa* with *E. nutans* preferred strongly by root voles (Liu, Wang & Liu 1991). Root vole populations in this area fluctuate only seasonally, with the lowest levels occurring in early spring; multiyear cycles are weak or absent (Jiang *et al*. 1991). The average population size across the study sites ranged from 70 to 170 voles ha^{-1} during the past 20 years, while in certain dense grassland sites, where grazing activities were limited and vegetation consisted mainly of *E. nutans,* the density reached to *c*. 400 voles ha^{-1} in late autumn (high level season, Jiang *et al*. 1991; Bian *et al*. 1994; Sun *et al*. 2002). The breeding season typically lasts from April to late October. Juveniles reach puberty and breeding age at *c*. 50 and 70 days, respectively (Liang *et al*. 1982). Thus, individuals born in spring normally start breeding within the year of birth. The dominant predators in this area are falcon *Falco tinnunculus*, buzzard *Bulteo hemilasius* and weasels *Mustela altaica*.

EXPERIMENTAL FACILITY

The experiment was carried out in eight 0·15-ha (50 × 30 m) outdoor enclosures. These were constructed using galvanized steel panels (1·5 m above and 0·5 m below-ground) and prevented mammalian predators from gaining entry. Avian predators were excluded with 3 × 3 cm grid wire mesh held aloft by a central pillar (10 × 250 cm) in each enclosure. Each enclosure was equipped with 60 laboratory-made wooden traps (Bian *et al*. 2011), spaced in a 5 × 5 m grids. Each trap was covered with a wooden sheet to protect from exposures to precipitation and temperature extremes. Prior to beginning the experiment, all enclosures were trapped for 2 weeks to remove resident small mammals. The traps were set between 7 am and 7 pm, baited with carrot, checked every 2 h and locked closed when trapping did not occur. Total of 12 voles were captured from the eight enclosures throughout the trapping period with 11 captured during the first week of trapping and one vole on the 9th day of trapping. Captured voles were removed to laboratory for other use. There were no other mammal species captured within any enclosures throughout the study period.

ESTABLISHMENT OF PARENTAL POPULATIONS AND LIVE-TRAPPING

One hundred and forty-four voles for each sex, 6 months of age or older, were used to establish parental populations. They were either F2 generations born in laboratory or captured as juveniles from previous year. Individuals were ear-marked before released into the enclosures. The parental populations were introduced into the enclosures in April 2012 at two density conditions. The low-density condition consisted of six adults per sex in each of the four enclosures and the high-density condition consisted of 30 adults per sex in each of another four enclosures. The initial body weights did not differ among voles in different enclosures ($F_{7,280} = 1{\cdot}72$, $P = 0{\cdot}103$). Live-trapping started after allowing animals to acclimate to the new environments for 2 weeks and lasted until late July.

Standard capture–record–recapture methods were used throughout the present study. Six trapping sessions were conducted; each consisted of 3 trapping days. The time intervals between two trapping sessions were *c*. 1 week. The traps were set between 7 am and 7 pm and were checked every 2 h on each trapping day. Following each capture, animal identification, sex, body mass and reproductive status (males, testes abdominal or scrotal; females, palpable embryos and enlarged teats barren of hair) were recorded, and the animal was then released back to the enclosures.

F1 offspring captured at 20–30 days of age (10–16 g, Liang *et al*. 1982) were removed to the laboratory and were ear-marked for subsequent offspring studies. They were maintained in the temperature-controlled colony (20 ± 2 °C) and singly housed in plastic cages with standard bedding and with pellet chow and water available *ad libitum*, under an 18/8 h light/dark cycle for 4–8 weeks until later use.

Faecal samples for faecal corticosterone metabolites (FCM) analysis were only collected from an adult trapped during the first two hr of trapping (07:00–09:00) and collected once from each animal in each trapping session. Thus, samples were not contaminated by any other intruders. Collected samples were then frozen with dry ice, transported to laboratory and stored in −20 °C freezer until analysis.

ESTABLISHMENT OF OFFSPRING POPULATIONS AND LIVE-TRAPPING

Subsequent establishment of offspring enclosures was performed *c*. 2 weeks after completion of parental population study. Prior to the start of the offspring experiment, the enclosures were trapped daily for two weeks to remove all small mammals within enclosures. The selected F1 offspring (50 days of age or older) from the two parental populations were each assigned into four enclosures with two for each of the two density treatments used in establishment of parental populations. Kin effect was minimized following more evenly distributions of offspring from enclosures in each parental source into each treatment groups. Maternally unstressed offspring (from low parental density source) assigned in low- and high-density populations were referred to as LL and LH, while maternally stressed offspring (from high parental density source) assigned in low- and high-density populations were referred to as HL and HH groups, respectively.

Live-trapping of offspring populations started 2 weeks after the establishment of offspring enclosures. Trapping was conducted for two seasons, one from 1 September to 23 October in 2012 and another from 8 March to 26 June in 2013. Five and eight trapping sessions were conducted in 2012 and 2013, respectively, with 3 trapping days for each session as described for parental populations. The time intervals between the two sessions were *c*. 10 days depending on the weather conditions. No parental individuals and un-marked F1 offspring were captured in any treatment groups throughout the two trapping seasons.

Offspring identification, sex, body mass and reproductive status were recorded from each capture, and faecal samples for both F1 offspring and recruits were collected and stored as previously described for parental populations.

FCM MEASUREMENT

FCM level reflects the level of circulating corticosterone that occurred 10–12 h previously in root voles (He *et al.* 2013), and FCM is primarily derived from plasma free corticosterone in rodents (Palme *et al.* 2005; Sheriff, Krebs & Boonstra 2010a).

Collected faecal samples were first lyophilized (Labconco, USA) for 14–18 h, and then ground into particles and homogenized in 0·5 mL NaOH solution (0·04 M). Extraction of FCM was performed by adding 5 mL of CH_2Cl_2 to the sample followed by sonication for 15 min (Pihl & Hau 2003) and centrifugation for 15 min at 3000 g. After centrifugation, 1 mL of solution was taken from the organic layer, diluted first with 3 mL CH_2Cl_2 and then mixed with 4 mL of mixed solution of sulphuric acid and ethanol (volume ratio, 7 : 3). The samples were then shaken for 2 min and left on table for 30 min before separation of the sulphuric acid layer for fluorescence detections. The fluorescence density in each sample was measured using a RF-540 IPC fluorometer (Shimadzu, Japan) at excitation and emission wavelengths of 470 and 520 nm, respectively, and FCM levels in each samples were calculated in reference to the fluorescence densities produced by varying concentrations of standard (Chen *et al.* 2012). We found that FCM levels in male and female voles did not differ significantly in each of the treatment groups. Thus, FCM levels in each treatment group were analysed following pooling the data from both sexes.

DATA COLLECTION

Population density

Population densities across trapping sessions in each enclosure of parental and offspring populations were estimated using the minimum number known alive (MNKA) method. For the parental populations, since F1 offspring were mostly removed from the enclosures on their initial captures, densities in parental enclosures were estimated with numbers of captured parents and thus representing only the parental densities across trapping sessions. For the offspring populations, population densities during 2002 trapping season were estimated with the numbers of offspring captured from two generations, F1 as original founders and F2 as recruits entering into populations. During 2013 trapping season, since F2 offspring born in 2012 also participated in reproduction, recruits captured during that season were either F2 from the original founders (F1) or F3 from F2 born in 2012. Therefore, offspring population densities during 2013 trapping season were estimated with the numbers of offspring captured from three generations, with F1 and F2 from 2012 as adults and F2 and F3 born during the trapping season as recruits.

Offspring reproductive condition and recruitment

Offspring reproductive condition in each group was evaluated using the proportion of reproductively active voles to the total numbers of adults captured for each sex in a trapping session. Recruitment rate was calculated as proportion of recruits captured in a trapping session to adult females captured in the second preceding session in each enclosure.

Overwinter survival

Offspring overwinter survival rate in each enclosure was calculated as proportion of F1 or F2 adults captured in 2013 to the numbers of F1 or F2 offspring captured in October 2012. The generation ratio in overwintered populations was calculated as proportions of overwintered F1 or F2 offspring to the total numbers of overwintered offspring (F1+F2) in each enclosure. Since only two F2 offspring were recruited in the HH group in 2012, we excluded F2 in the statistical analysis of overwinter survival rates from the HH group.

STATISTICAL ANALYSIS

Parental or offspring population densities (Poisson distribution) and FCM levels (linear model), and proportion of reproductive individuals and overwinter survival (Binomial distribution) and recruitment rates (Linear model), were analysed using generalized linear mixed model (GLMM) in SPSS v.19 (IBM, Armonk, NY, USA) with Log/Logit link functions. For repeated measures in GLMM, since response variables may be correlated to observational units (Enclosures) at different time points (trapping session), we first carried out a comparison of candidate models with various covariance structures using Akaike information criterion, corrected (AICc). The model with the smallest AICc value was then selected and subsequently simplified by dropping the insignificant ($P > 0·05$) interactions during the analysis. *Post hoc* comparisons for significant treatment effects were followed using the sequential Bonferroni *post hoc* procedure.

The relationship between maternal GC activity and offspring quality in the parental populations was analysed with general least-square regression, using mean female FCM level and F1 offspring body mass at sexual maturity from each enclosure as independent and dependent variables, respectively. In the offspring populations, the correlations of offspring GC activity with the proportion of reproductive individuals and recruitment were analysed using mean adult female FCM levels per enclosure in each of the two trapping seasons (2012 and 2013) as independent variables and the corresponding rates of reproductively active females and recruitment for that enclosure and season as dependent variables.

Results

PARENTAL DENSITY AND FCM LEVELS ACROSS TRAPPING SESSIONS

Parental densities in the high-density parental enclosures declined progressively following the trapping sessions while the densities in the low-density enclosures remained relatively stable (Fig. 1a). We found a significant effects of treatment ($F_{1,36} = 684·27$, $P < 0·001$), time ($F_{5,36} = 12·63$, $P < 0·001$) and treatment × time interaction ($F_{5,36} = 4·21$, $P = 0·004$). The decline in the high-density enclosures was more prominent in male voles. The mean estimated numbers of male and female parents at the end of the experiment were $5·24 \pm 0·82$ and $14·50 \pm 1·67$ per enclosure in high density (initial density, 30 per sex per enclosure) and $2·50 \pm 0·65$ and $3·00 \pm 1·11$ per enclosure in low-density (initial density, 6 per sex per enclosure) parental populations, respectively.

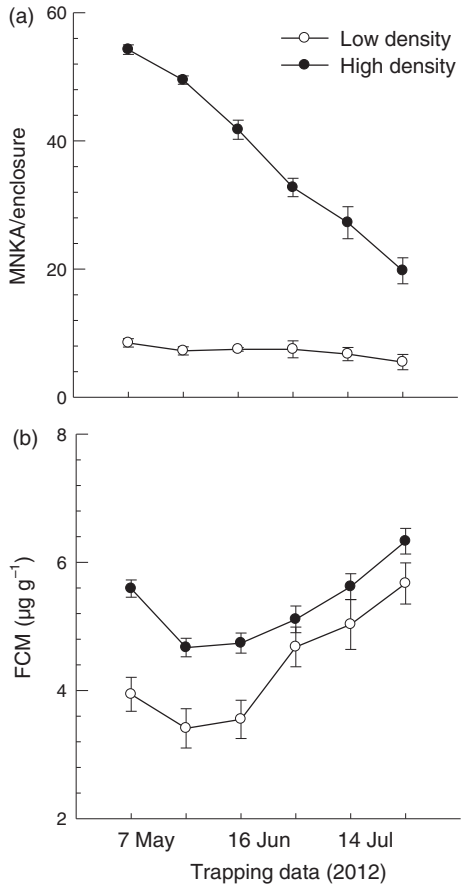

Fig. 1. Parental densities (a) and FCM levels (b) across live-trapping sessions in high- and low-density parental populations. Parental density was estimated as minimum number known to be alive (MNKA) in each enclosure. Data from four enclosures in each of the two density groups were expressed as mean ± SE. $N = 123$ and 423 for FCM measurements in low- and high-density groups, respectively. [Correction added on 8 January 2015 after first online publication: MNAK changed to MNKA.]

Mean parental FCM levels in the high- and low-density populations were 5·26 ± 0·07 and 4·28 ± 0·14 μg g^{-1}, respectively. Parental FCM levels in the high-density population were elevated from the time of the first trapping session and sustained at elevated levels across all the trapping sessions (Fig. 1b). We found significant effects of treatment ($F_{1,539} = 4·78$, $P = 0·029$) and time ($F_{5,539} = 23·17$, $P < 0·001$), but not treatment × time ($F_{5,534} = 1·72$, $P = 0·127$).

The mean body masses at sexual maturity in male and female F1 offspring were 23·12 ± 0·46 and 22·69 ± 0·51 g from the high density and 26·67 ± 0·57 and 24·88 ± 0·34 g from the low-density populations, respectively. We found significant effects of treatment ($F_{1,232} = 14·34$, $P < 0·001$) and gender ($F_{1,232} = 4·86$, $P = 0·028$), but not of treatment × gender interaction ($F_{1,231} = 0·38$, $P = 0·537$).

Regression analysis of mean female FCM level with F1 offspring body mass at sexual maturity per enclosure revealed a significant, negative correlation between maternal GC activity with F1 offspring quality ($r^2 = 0·57$; $y_{\text{body mass}} = 40·36 - 0·382x_{\text{FCM}}$; $F_{1,6} = 7·90$, $P = 0·031$).

OFFSPRING DENSITY AND FCM LEVELS ACROSS TRAPPING SESSIONS

Offspring populations fluctuated differently in the four treatment groups during both 2012 (treatment, $F_{3,20} = 8·50$, $P = 0·001$; time, $F_{4,20} = 3·68$, $P = 0·021$; treatment × time interaction, $F_{12,20} = 5·51$, $P < 0·001$; Fig. 2a) and 2013 (treatment, $F_{3,27} = 11·14$, $P < 0·001$; time, $F_{7,27} = 25·08$, $P < 0·001$; interaction, $F_{21,27} = 3·20$, $P = 0·003$; Fig. 2b) trapping seasons. Population density in LL group increased progressively from an initial density of 12 per enclosure across the entire trapping season in 2012 and reached the similar levels seen in LH and HH groups (34·50 ± 8·50, 57·00 ± 15·00 and 39·50 ± 0·50 per enclosure in LL, LH and HH groups, respectively) at the last trapping session. During the 2013 season, population in LL, LH and HH groups were maintained at steady levels until 18 May and increased rapidly during the last tapping month (Initial density: LL, 26·00 ± 12·00; LH, 36·50 ± 10·50; HH, 40·00 ± 15·56 per enclosure. Final density: LL, 64·00 ± 5·00; LH, 75·50 ± 24·50; HH, 87·50 ± 26·50 per enclosure). In contrast, offspring population in HL group was sustained at its initial low level (11·50 ± 3·50) throughout the two trapping seasons (11·50 ± 1·50 at the last trapping session in 2013).

Mean offspring FCM levels were 1·94 ± 0·03, 3·08 ± 0·03, 3·32 ± 0·06 and 3·73 ± 0·03 μg g^{-1} in 2012 and 2·04 ± 0·03, 3·28 ± 0·03, 4·13 ± 0·05 and 3·74 ± 0·03 μg g^{-1} in 2013 in LL, LH, HL and HH groups, respectively. Offspring FCM levels in LH, HL and HH groups significantly increased in 2012 from initial levels of 2·75 ± 0·05, 2·59 ± 0·09, and 3·29 ± 0·05 μg g^{-1} to 3·43 ± 0·07, 4·18 ± 0·13 and 3·89 ± 0·06 μg g^{-1} at the end of the trapping sessions (Fig. 2c and d). FCM levels remained high throughout the 2013 trapping sessions (Fig. 2d). In contrast, FCM in LL group were remained at low steady levels from initial level of 1·79 ± 0·084 to 1·96 ± 0·17 at the end of the trapping sessions (Fig. 2c and d). We found significant effects of treatment ($F_{3,518} = 728·08$, $P < 0·001$; $F_{3,353} = 274·72$, $P < 0·001$) and time ($F_{4,338} = 89·77$, $P < 0·001$; $F_{5,353} = 7·74$, $P < 0·001$) for both 2012 and 2013 seasons, and a significant effect of treatment × time interaction for 2012 ($F_{12,518} = 5·23$, $P < 0·001$), but not for 2013 ($F_{15,338} = 0·67$, $P = 0·818$). *Post hoc* analysis indicated that FCM in LH, HL and HH groups were significantly higher than in LL group across all trapping sessions in both seasons ($P < 0·05$).

OFFSPRING REPRODUCTIVE CONDITION, RECRUITMENT AND OVERWINTER SURVIVAL

Mean proportions of reproductively active male and female offspring varied significantly among the treatment

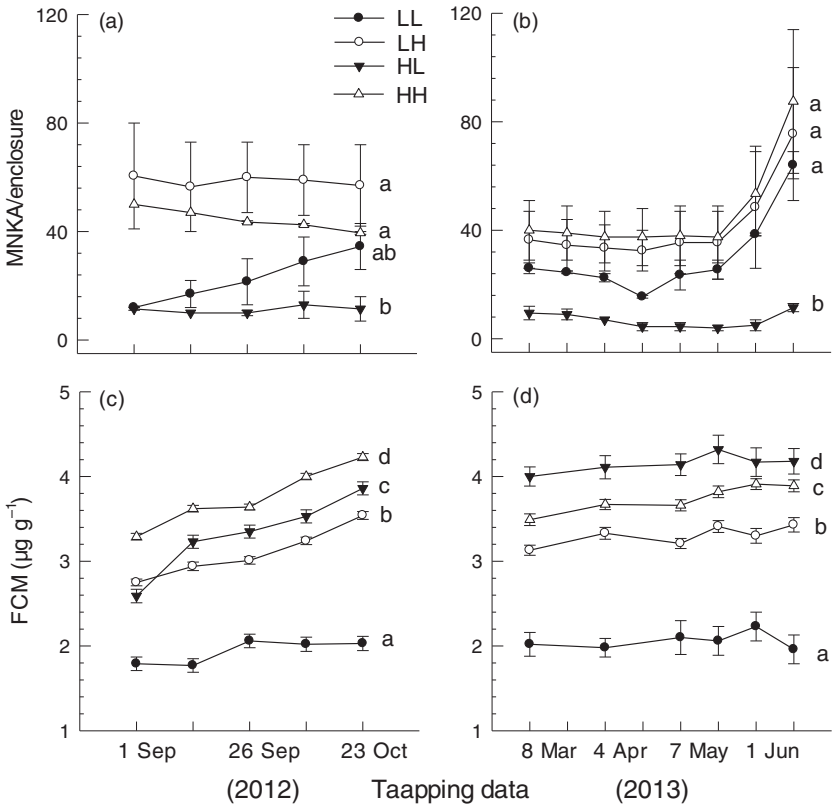

Fig. 2. Fluctuations of offspring densities (a, b) and FCM levels (c, d) across trapping sessions during 2012 and 2013 seasons in maternally stressed, low (HL) and high (HH) and maternally unstressed, low (LL) and high (LH) density offspring enclosures. Data were expressed as mean ± SE for each group. Group levels that share same small letters indicate no significant difference. N = 84, 319, 94 and 403 for FCM measurements in LL, LH, HL and HH group, respectively.

groups (0·66 ± 0·17, 0·24 ± 0·07, 0·04 ± 0·09 and 0·04 ± 0·03 in male and 0·69 ± 0·02, 0·17 ± 0·07, 0·23 ± 0·13 and 0·02 ± 0·01 in female in LL, LH, HL and HH, respectively) in 2012. High-density exposure either during the maternal period or during adulthood resulted in a significant decrease in the proportions of reproductively active individuals in both males and females, with a more prominent decrease found following exposure during maternal period (Fig. 3a and b). We found significant effects of treatment in both sexes (male, $F_{3,36}$ = 10·29, P < 0·001; female, $F_{3,36}$ = 24·90, P < 0·001), with the greatest number of reproductively active individuals found in the LL group. In the 2013 season, no statistical differences were found in either sex among the four treatment groups in 2013 (male, $F_{3,55}$ = 0·11, P = 0·954; female, $F_{3,44}$ = 1·71, P = 0·179).

Mean recruitment rates in LL, LH, HL and HH groups were 1·16 ± 0·30, 0·20 ± 0·06, 0·20 ± 0·13 and 0·07 ± 0·07 in 2012 and 2·02 ± 0·49, 1·30 ± 0·57, 0·75 ± 0·40 and 1·13 ± 0·45 in 2013 trapping seasons, respectively. We found significant treatment effects for both 2012 ($F_{3,36}$ = 16·90, P < 0·001) and 2013 ($F_{3,28}$ = 211·49, P < 0·001) trapping seasons, with the highest level seen in LL group in both trapping seasons and the lowest level seen in HH and HL groups in 2012 and 2013, respectively (P < 0·05, Fig. 4).

Overwinter survival rates of F1 and F2 offspring varied significantly among the treatment groups (Fig. 5a). LL group showed the lowest rate in F1 (0·26 ± 0·01) and the highest rate in F2 (0·76 ± 0·02) offspring, respectively, while the opposite was observed in HL (F1, 0·80 ± 0·19; F2, 0·40 ± 0·01) group. We found significant effects of treatment in both F1 and F2 populations (F1, $F_{3,11}$ = 243·06, P < 0·001; F2, $F_{2,6}$ = 76·84, P < 0·001). The generation ratio in overwintered populations was significantly different among the four groups ($F_{3,4}$ = 8·06, P = 0·036). The ratio of F2 offspring in the overwintered population in LL group was significantly higher than in other three groups (P < 0·05, Fig. 5b).

Correlation analysis using adult female FCM per enclosure in each trapping season as an independent variable indicated that adult female offspring FCM levels were negatively correlated to both proportion of reproductively active females (r^2 = 0·485, $y_{\text{Proportion of reproductive individuals}} = 1{\cdot}08 - 0{\cdot}21x_{\text{FCM}}$, $F_{1,14}$ = 13·18, P = 0·003, Fig. 6a) and recruitment rates (r^2 = 0·256, $y_{\text{recruitment rate}} = 2{\cdot}28 - 0{\cdot}46x_{\text{FCM}}$, $F_{1,14}$ = 8·81, P = 0·046; Fig. 6b) in offspring populations.

Discussion

Within the well controlled, predator excluded enclosures, we found that greater population density, whether during the maternal period or adulthood in root vole was associated with an activation of individual stress axis (Figs 1 and 2). The elevated FCM levels in HL and HH groups suggest a long-lasting effect of maternal density on offspring GC activity (Fig. 2c and d). More importantly, we

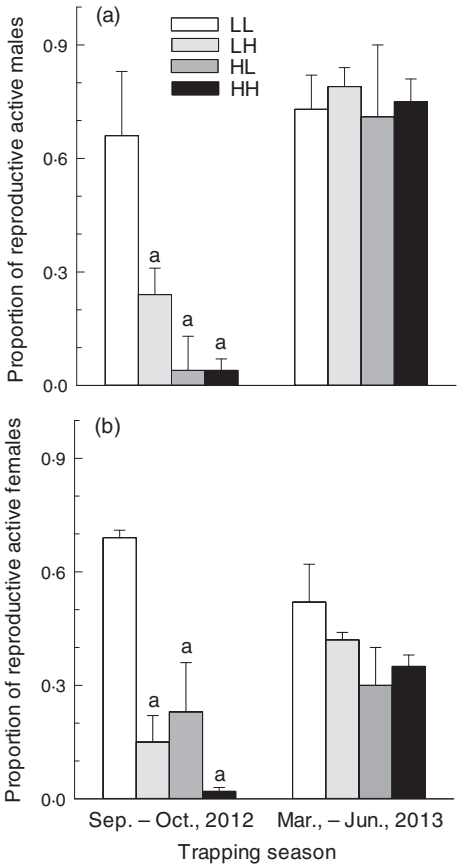

Fig. 3. Mean (±SE) proportions of reproductively active males (a) and females (b) across two trapping seasons (2012 and 2013) in LL, LH, HL and HH offspring populations. Data obtained from 2012 trapping season represent proportions of reproductive individuals of F1 founders and from 2013 represent the proportions of reproductive individuals of overwintered F1 founders and F2 recruits in each treatment group. a, $P < 0{\cdot}05$ vs. LL group in the trapping season.

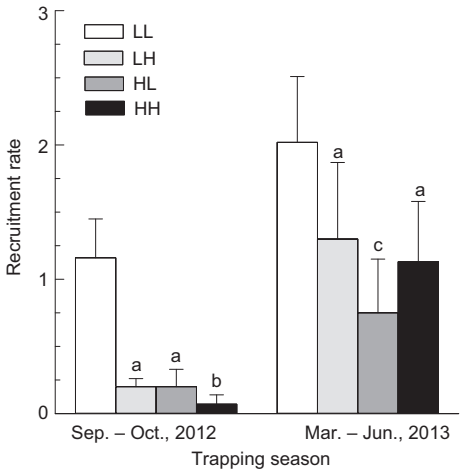

Fig. 4. Mean (±SE) recruitment rates across two trapping seasons in LL, LH, HL and HH offspring populations. Data obtained from 2012 trapping season represent the recruitment rate of juveniles (F2) from F1 founders and from 2013 represent recruitment rate of juveniles (F2+F3) from overwintered founders and recruits in each treatment group. a, $P < 0{\cdot}05$ vs. LL group; b, $P < 0{\cdot}05$ vs. LL and LH groups; c, $P < 0{\cdot}05$ vs. LL, LH and HH groups in the season.

found that maternal density stress resulted in a significant decrease in offspring reproduction, regardless of the offspring adult density (Figs 3 and 4). These findings suggest an important role of maternal environment in determining offspring phenotypes that are relevant to the population dynamics in wild small mammals and indicate that maternal stress system is likely involved in mediation of such processes.

MATERNAL POPULATION DENSITY ON OFFSPRING REPRODUCTION AND POPULATION FLUCTUATIONS

One of the important findings from the present study is an impaired response of reproductive system in maternally density-stressed F1 offspring. We found that while the reproduction suppression occurred only in LH populations of maternally unstressed offspring, it occurred in both HL and HH populations. Moreover, we found that the proportions of reproductively active F1 adults and the recruitment rates in HL and HH were significantly lower than in LL and LH populations, respectively (Figs 3 and 4). As a consequence of such suppression, offspring populations were maintained relatively stable across the first trapping season in density-stressed populations (Fig. 2a) and the proportions of F1 offspring in overwintered adults were significantly higher in density-stressed than in density-unstressed populations (Fig. 5b). These findings are in general accordance with those from snowshoe hare studies in which predation was used as a stressor (Sheriff, Krebs & Boonstra 2010b). The present findings are somewhat in contrast to those from the transplant field study by Ergon and colleagues, showing that neither animals' growth rates nor reproduction traits did display their source characters, due to the overriding influence of the immediate environment in which animal had been introduced (Ergon, Lambin & Stenseth 2001). In that study, the authors applied body mass only as measure of density stress without detailed recording developmental history in transplanted animals. Thus, the extent to which the transplants were stressed is not well clear.

Population density has been suggested as a self-regulatory factor in population stabilization (Ostfeld, Canham & Pugh 1993; Inchausti *et al.* 2009) and thus tending to shift the population size to a level optimal for the population. Our present findings from maternally unstressed but not stressed offspring are in agreement with this hypothesis. Despite the vast difference at the initial setup, population size in the LH and LL groups tended to move to the same level throughout the time during the first trapping season and reached to this level at the beginning of the second trapping season (Fig. 2a and b). This self-regulation is likely achieved by the decreased survival rate and increased breeding effort in LH and LL enclosures,

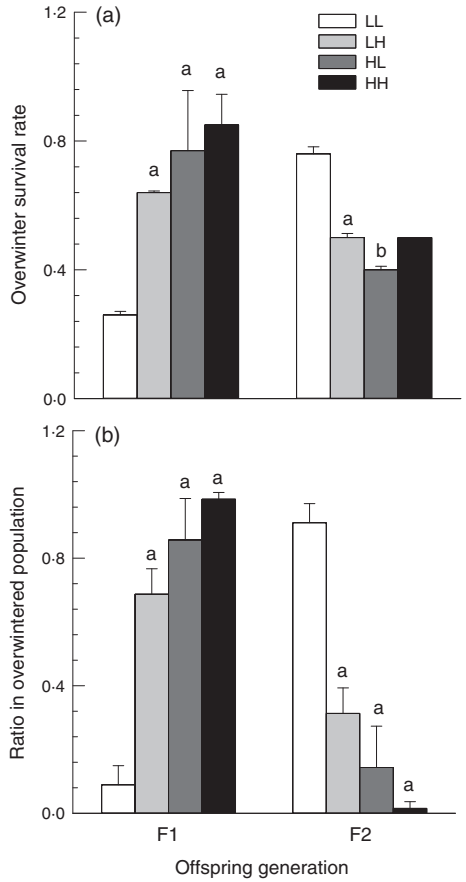

Fig. 5. Overwinter survival rates (Mean ± SE) of F1 and F2 offspring (a) and generation ratio (Mean ± SE) in overwintered populations (b) in offspring populations. Since only two F2 voles were recruited into the HH population in 2012 trapping session, we excluded this group in the rate analysis. a, $P < 0{\cdot}05$ vs. LL group; b, $P < 0{\cdot}05$ vs. LL and LH groups.

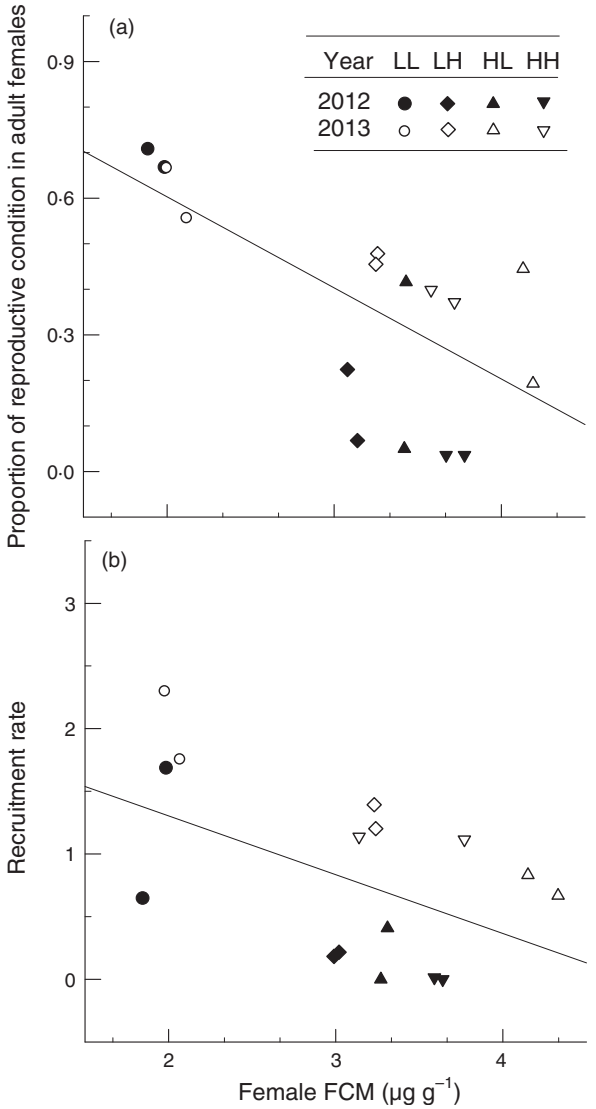

Fig. 6. Correlation analysis of female offspring GC activity with proportion of reproductive individuals (a) and recruitment rate (b) in each offspring enclosure during 2012 and 2013 trapping seasons. Mean adult female FCM levels per enclosure in each of the two trapping seasons were used as independent variables and the corresponding proportion of reproductively active females and recruitment rate for that enclosure and season as dependent variables. The solid line in each graph represents the general least-squares regression equation generated from analysis. Both offspring proportion of reproductive individuals and recruitment rate were negatively correlated to adult female FCM levels in the enclosure ($r^2 = 0{\cdot}485$, $y_{\text{Proportion of reproductive individuals}} = 1{\cdot}08 - 0{\cdot}21x_{\text{FCM}}$, $P < 0{\cdot}05$; $r^2 = 0{\cdot}256$, $y_{\text{recruitment rate}} = 2{\cdot}28 - 0{\cdot}46x_{\text{FCM}}$, $P < 0{\cdot}05$).

respectively, since recruitment rate was found significantly higher in LL than LH groups (Fig. 4). In contrast, the population levels in the HL and HH groups were both remained relatively steady, indicating an impaired capacity in maternally stressed offspring in response to immediate environment (Fig. 2a and b) and this in turn resulted in a sustained low level of population in HL group throughout the entire testing period.

The sustained low population levels in the HL group indicate a possible involvement of intrinsic physiological state in generating delayed density-dependent effects. Delayed density-dependent processes are considered as major mechanisms in generating population cycles (Beckerman *et al.* 2002) and specialist predator–prey interaction has been commonly recognized as a primary cause generating these processes (Stenseth 1999). In the present study, however, the enclosures were well isolated from common vole predators, and thus predators would have never encountered in the populations. It is also unlikely that bottom-up processes had any significant effect on the low levels in this group, since above-ground plant biomasses would have been more abundant in HL than in other enclosures and thus resource competition would expect to be more intense in high-density offspring populations. Thus, considering also the extremely low levels of overwintered recruits in HL populations, it is reasonable to believe that the sustained low populations in this group result from the long-lasting reproduction suppression in F1 offspring induced by maternal density stress.

MATERNAL STRESS SYSTEM AS A POTENTIAL MEDIATOR IN TRANSLATING MATERNAL ENVIRONMENTS INTO OFFSPRING PHENOTYPES

Stress response to environmental challenge is well recognized as a biological costing process. This cost can be an alteration in individual behavioural phenotypes or in breeding efforts, and to the worst scenario, an expense of individual life (Boonstra 2004). Many of these alterations are also observed in animals with artificially elevated GC levels (see review by Johnson *et al.* 1992). Laboratory studies indicate that stress during pregnancy and lactation results in lifelong programming of the offspring stress axis including an elevated GC level that resemble that of their mothers (Welberg & Seckl 2001). However, the biological relevance of these studies to wild-living mammals has not been broadly explored. In snowshoe hare populations, Sheriff *et al.* showed that predation-induced maternal FCM elevations were not only related to maternal reproduction suppression but were also echoed in their offspring, and this occurred at population-wide levels (Sheriff, Krebs & Boonstra 2009, 2010b). The present findings in FCM levels and reproduction traits in HL populations are the first evidence indicating that such maternal programming effect persists even in an environmental context that are substantially different from that offspring experienced during the maternal period. Either hyperactivity in stress axis or chronic exposure to high GC levels has been shown to suppress functions of the hypothalamic–pituitary–gonadal axis in laboratory animals (see review by Wingfield & Sapolsky 2003). Thus, the reproduction suppression seen in stressed offspring is likely a consequence of elevated GC activity in these animals and the stress system is likely one of the physiological systems that mediates the translation of maternal environment into offspring life-history traits or transmits phenotype transgenerationally (Love, McGowan & Sheriff 2013; Sheriff & Love 2013).

While the present study has demonstrated a role of maternal stress system in density-induced alterations in offspring phenotypes, the role of this system on offspring is not limit to the effects of intrinsic factors. An increase in the number of predators, a risk of predation, or even a perceived risk of predation can increase maternal GCs (Boonstra *et al.* 1998; Sheriff, Krebs & Boonstra 2009, 2011; Travers *et al.* 2010; Zanette *et al.* 2011). Increases in maternal GCs can also occur directly via reductions in the quantity, quality and predictability of resources (Jeanniard du Dot, Rosen & Trites 2009), or indirectly via a reduction in maternal condition (Meylan *et al.* 2002; Love *et al.* 2005). Interactions between resources and predation risk have been shown acting synergistically to increase GCs (Clinchy *et al.* 2004; Sheriff, Krebs & Boonstra 2010b). Therefore, maternal stress system may act as a common interface at which both intrinsic and extrinsic factors interact and influence offspring phenotypes.

MATERNAL DENSITY-INDUCED REPRODUCTION SUPPRESSION VIEWED FROM AN ADAPTATION PERSPECTIVE

One of the most intriguing properties of maternal effects is that they transmit environmental influences through time (Bernardo 1996); consequently, their duration and hence, their impact upon offspring and offspring population can vary not just in magnitude but in the total time over which the effects are expressed. From an adaptation point of view, maternal effects reflect at which degree they predict the environmental circumstances that offspring are likely to experience in adulthood. Therefore, evaluations of the persistency, as well as the context-contingency of maternal effects are essential in terms of understanding the process that maternal environments influence offspring individual fitness and offspring population dynamics. For individuals in a population, reproduction suppression is believed to increase the likelihood of survival to the next breeding season and thus was typically interpreted as an adaptive reproductive strategy. The significant higher rate of F1 overwinter survival and higher proportions of F1 offspring in overwintered populations of density-induced maternally stressed groups (HL and HH, Fig. 5), and the significant lower recruitment in the density-stressed groups in the present study are in agreement with this hypothesis. At a population level, however, such suppression may have different consequences under different population backgrounds. While in the LH and HH groups, such suppression could be explained as an adaptive response to the high density in the populations, the reproductive suppression seen in F1 offspring in the HL group would be a biological costly consequence due to great cost of losing reproductive opportunity in a more favourable environment, and thus maladaptive. Therefore, our present findings support the notion that the adaptive potential of phenotype variations induced by maternal effect should be viewed within an ecologically relevant or a life-history optimization framework (Love, McGowan & Sheriff 2013; Sheriff & Love 2013).

It is also necessary to note that vole populations subjected to different externally driven dynamics may have evolved different life-history tactics and that the present study was conducted in root voles originated from seasonally fluctuated, non-cyclic populations. Thus, the effects of maternal density on offspring found in the present study may have differential impact on offspring from populations with different fluctuation characteristics. Several studies have shown that voles from cyclic populations are more sensitive to social or density-related factors while voles from non-cyclic populations are more sensitive to external factors such as predation (Gustafsson, Andersson & Nyholm 1983; Bondrup-Nielsen & Ims 1986). Thus, it is reasonable to speculate that the effects induced by maternal population density may have even more profound impact on voles from cyclic populations than seen in the present study.

SUMMARY AND FUTURE DIRECTIONS

The present study provides the first evidence in the controlled offspring populations that maternal stress may significantly impair offspring reproductive fitness when there is a mismatch between the maternal and offspring environment. The present study suggests that maternally derived stress might be a common mechanism through which most ecological factors modulate offspring population dynamics. Future studies aiming at verifications of the sufficiency of stress system in this modulation would be necessary before definite conclusion can be reached.

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Data accessibility

Data available from the Dryad Digital Repository: http://dx.doi.org/10.5061/dryad.c7885 (Bian *et al*. 2014).

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